In the United States Court of Federal Claims

OFFICE OF SPECIAL MASTERS
No. 22-335V

CHARLES TAYLOR, Petitioner, v. SECRETARY OF HEALTH AND HUMAN SERVICES, Respondent.	Chief Special Master Corcoran Filed: March 25, 2025

Elizabeth K. Abramson, mctlaw, Washington, DC, for Petitioner.

Nina Y. Ren, U.S. Department of Justice, Washington, DC, for Respondent.

RULING ON ENTITLEMENT[1]

On March 28, 2022, Charles Taylor filed a petition for compensation under the National Vaccine Injury Compensation Program (the "Vaccine Program").[2] *See* Petition (ECF No. 1). Petitioner alleges that an influenza ("flu") vaccine administered to him on October 5, 2020, caused him to experience Guillain-Barré syndrome ("GBS"). *Id.*

The elements of a Table flu vaccine-GBS claim are met by the records filed in this matter. But because Respondent has argued that evidence supported an alternative cause for Petitioner's injury, I proposed that the parties brief whether Respondent could carry his shifted burden of proof. *See* Petitioner's Motion, dated Aug. 9, 2024 (ECF No. 50-1) ("Mot."); Respondent's Opposition,

[1] "Under Vaccine Rule 18(b), each party has fourteen (14) days within which to request redaction "of any information furnished by that party: (1) that is a trade secret or commercial or financial in substance and is privileged or confidential; or (2) that includes medical files or similar files, the disclosure of which would constitute a clearly unwarranted invasion of privacy." Vaccine Rule 18(b). Otherwise, the whole Ruling will be available to the public in its present form. *Id.*"

[2] The Vaccine Program comprises Part 2 of the National Childhood Vaccine Injury Act of 1986, Pub. L. No. 99-660, 100 Stat. 3758, codified as amended at 42 U.S.C. §§ 300aa-10 through 34 (2012) [hereinafter "Vaccine Act" or "the Act"]. Individual section references hereafter will be to § 300aa of the Act (but will omit that statutory prefix).

dated Sep. 9, 2024 (ECF No. 53) ("Opp."); Petitioner's Reply, dated Sep. 16, 2024 (ECF No. 54) ("Reply").

Now, having reviewed the above plus the filed medical records, expert reports, and associated literature, I hereby find that Respondent has not preponderantly demonstrated that a "factor unrelated" – namely a cytomegalovirus ("CMV") infection[3] – was the more likely, and sole, explanation for Petitioner's GBS. Accordingly, entitlement is established.

I. Factual Background

Mr. Taylor has a pre-vaccination history of migraine headaches, and had experienced several tick bites in 2019 and 2020 requiring prophylactic treatment with Doxycycline. Ex. 2 at 49, 51-54, 57-60.[4] He received the flu vaccine in question on October 5, 2020, in New York City. Ex. 1 at 1. There is no record evidence of any immediate reaction or symptoms close-in-time to vaccination.

A week later (October 12th), Petitioner sought treatment for reported left shoulder pain, which he said had started after working on his cabin. Ex. 5 at 5-10. Exam revealed no acute injuries or abnormalities, and no neurological deficits were observed on physical examination. *Id.* at 9. Also, no reference to the vaccination was made (or to any neurologic-like symptoms later reported). Petitioner was diagnosed with left shoulder tendonitis and discharged after receiving a Toradol (nonsteroidal) injection. *Id.* at 10.

Ten days after vaccination (October 15, 2020), Petitioner took himself to the Columbia University Student Health Center complaining of fatigue, headaches, tingling in his hands and feet, and generalized weakness, beginning five days before (meaning October 10th). Ex. 2 at 43. He also confirmed a history of tick bites, along with the fact that he had been experiencing headaches (distinguishable from migraines) for up to two weeks (which would predate vaccination). *Id.* at 43-44. Blood testing performed that day yielded unremarkable results, except for slightly elevated liver function tests ("LFTs") (AST: 48 IU/L and ALT: 61 IU/L). *Id.* at 41, 84. And another neurologic test resulted in normal findings. *Id.* at 44-45. Petitioner was assessed with "unspecified disturbances of skin sensation." *Id*. at 45.

Then, on the evening of October 16th, Petitioner went to an emergency room, again reporting left shoulder pain (which he associated with heavy chores and lifting his daughter) and limited range of motion, with an initial exam confirming his complaints (but identifying no neurologic concerns). Ex. 3 at 7-8. He was assessed with a probable rotator cuff injury and received

[3] CMV is a common, incurable virus that is part of the herpes family, and can linger in the body even after the infection's resolution. *Clinical Overview of CMV and Congenital CMV*, Center for Disease Control, https://www.cdc.gov/cytomegalovirus/hcp/clinical-overview/index.html (last visited Feb. 6, 2025).

[4] Although Petitioner reported on several occasions to treaters that he had experienced the tick bites (perhaps in the belief they could have played some role in his subsequent injury), the parties seem to concur that such bites do not likely explain his GBS, and I therefore do not include discussion of them as potentially causal in this Decision.

a steroid injection prior to his discharge. *Id.* at 8, 18. Petitioner's shoulder concerns persisted (to the point where he felt unable to move his arm), however, and the next day (October 17th) he returned to the Student Health Center for additional treatment. Ex. 2 at 35-36. Petitioner was advised to seek orthopedic assistance and was urged to otherwise continue with self-help treatments, supplemented by some pain-oriented prescriptions. *Id.* at 36.

Petitioner thereafter continued to experience shoulder pain-related issues, but also began expressing concerns about other symptoms that could be considered more neurologic in character. For example, on October 19, 2020, Petitioner contacted treaters about his previously-reported hand and feet paresthesia, plus worsening weakness, and questioned whether a neurologic consult was needed in addition to further orthopedic evaluations. Ex. 2 at 27, 30. He also reported difficulty walking up stairs and getting out of bed, and trouble with temperature regulation, leading him to question whether his condition had a neurologic explanation. *Id.* at 33 (Petitioner informing treater by email that "this sounds like peripheral neuropathy").

Petitioner took himself to New York Presbyterian Hospital's emergency department on October 21, 2020, complaining of pain and weakness in his arms and neck, plus tingling in his hands and feet, and leg weakness that inhibited his movement. Ex. 4 at 35. Petitioner was admitted to the hospital for further evaluation, including a neurology consult, and additional testing was ordered. *Id.* In the process of his intake, Petitioner fell down and remained prone until he was found by treaters, leading the hospital to take extra precautions with him going forward. *Id.* at 38.

After admission, Petitioner underwent an initial neurology consultation with Dr. Christoforos Koumas. Ex. 4 at 69-72. Dr. Koumas's assessment noted Petitioner's recent history of numbness and hand/foot tingling plus weakness, adding that (in addition to the previously-treated left shoulder pain) Petitioner had also begun to experience comparable *right* shoulder pain the day before admission (making him wonder if numbness was ascending). *Id.* at 69. And Dr. Koumas recorded Petitioner's complaints of generalized weakness and ambulation impacts, as well as the fact that Petitioner denied other symptoms or infections beyond "several tick bites this year." *Id.*

Petitioner's neurologic exam while hospitalized now confirmed diminished strength and reflexes, plus gait abnormalities. Ex. 4 at 70-71. The initial diagnostic differential included peripheral neuropathy, radiculoneuropathy, or cervical cord pathology, although more workup was deemed necessary before a particular etiology could be embraced. *Id.* at 72.

Petitioner underwent a more comprehensive neurologic evaluation on October 22, 2020, when two neurologists (other than Dr. Koumas) saw him. Ex. 4 at 38-45. In setting down the medical history, some additional details were added, such as (a) progression of Petitioner's paresthesia from fingertips and toes upward, (b) temperature regulation difficulties, and (c) fatigue that not only limited Petitioner's exercise ability but made it hard to engage in mundane activities of daily living. *Id.* at 39. But it was also stated that he had previously experienced episodes of

"incapacitating back pain" that would cause him to require a day of complete rest before resumption of activities, and Petitioner's spouse speculated that his current presentation was qualitatively similar. *Id.* The neurologic treaters further noted that Petitioner denied any pre-onset illness or infections (including ones he might have been exposed to via his infant daughter), and that he had received the flu vaccine earlier that month (plus the aforementioned tick bites that same year). *Id.* at 40.

Thereafter, treaters expressed greater confidence in a GBS diagnosis, which was supported by additional testing evidence. Additional neurologic exams continued to reveal Petitioner's weakness, diminished reflexes, and other comparable symptoms consistent with his presentation and primary complaints. Ex. 4 at 42. A lumbar puncture and cerebral spinal fluid ("CSF") analysis (performed on October 22, 2020), revealed elevated proteins consistent with GBS (and did not identify possible pathogenic explanations). *Id.* at 45, 76, 128–29. An electromyogram and nerve conduction study were also consistent with GBS (although not specific for it, as the results did not demonstrate the presence of demyelination). *Id.* at 28, 49. Treaters proposed Petitioner receive a course of intravenous immunoglobulin ("IVIg")[5] and neuropathic pain medications – treatments common in the setting of GBS. *Id.* at 38, 158.

At the same time, however, one set of testing revealed results relevant to the dispute herein regarding a potential etiology for Petitioner's GBS. Testing performed on Petitioner around October 21, 2020, revealed the presence of a recent, active CMV infection. Ex. 4 at 49 ("CMV quantitative PCR from blood returned at 990, serum IgG and IgM[6] were also positive"), 126. A "curbside"[7] infectious disease consult was performed on October 23, 2020, to discuss the test results. The infectious disease team opined that the positive results were more likely reflective of a latent CMV reactivation infection, secondary to Petitioner's GBS, than evidence of an active infection, and treatment was not deemed necessary in the context of ameliorating Petitioner's

[5] "Intravenous Immunoglobulin (IVIG)" is defined as "[a] therap[y] prepared from a pool of immunoglobulins (antibodies) from the plasma of thousands of healthy donors. Immunoglobulins are made by the immune system of healthy people for the purpose of fighting infections…IVIG/SCIG work in different ways to prevent the body from attacking itself and to decrease several types of inflammation in the body." IVIG, https://rheumatology.org/patients/intravenous-immunoglobulin-ivig (last visited Mar. 24, 2025).

[6] IgG and IgM are types of immunoglobulins, which are also referred to as antibodies. Antibodies are proteins that the immune system makes to fight germs. IgG antibodies, which are present in all body fluids, are very important for fighting bacteria and viruses. The body keeps a "blueprint" of all the IgG antibodies that have been created - so if a person is exposed to the same germs again, the immune system can quickly make more antibodies. IgM antibodies, which are found in the blood and lymph fluid, are the first antibodies the body makes after it is exposed to germs. They provide short-term protection while the body makes other antibodies. "Immunoglobulins Blood Test," *Medline Plus*, https://medlineplus.gov/lab-tests/immunoglobulins-blood-test/ (last visited Mar. 6, 2025).

[7] According to Dr. Collins, a curbside consultation involves "a casual request for information in which the consulting service does not review the chart or examine the patient" but "[i]nstead. . . are given a very brief summary of a particular question the team has." First Collins Rep. at 4.

immediate symptoms. *Id.* at 49 (CMV infection "[u]nlikely to be contributory to the current picture"), 93.

After receiving a three-day course of IVIg, Petitioner was discharged on October 25, 2020, with a diagnosis of GBS "likely in the setting of recent flu vaccine." Ex. 4 at 47, 49. He continued to experience common post-treatment GBS sequelae thereafter. Ex. 2 at 19. However, Petitioner made slow progress in recovering. *Id.* at 15. By February 2021, he was still experiencing some range of motion limitations plus weakness and numbness, and was advised to expect symptoms to linger for six months or so from onset (meaning through spring 2021). Ex. 2 at 11-12. By April 2021, Petitioner felt he was "about 70% back to baseline." Ex. 4 at 12. He continued to bear the diagnosis of GBS, AIDP (acute inflammatory demyelinating polyneuropathy) variant. *Id.*at 25.

II. Expert Reports[8]

A. *Respondent's Expert – Kathleen L. Collins, M.D., Ph.D.*

Dr. Collins, an infectious disease specialist and medical school professor, offered two written reports in this case for Respondent. Report, dated July 1, 2023, filed as Ex. A (ECF No. 34-1) ("First Collins Rep."); Report, dated Apr. 23, 2024, filed as Ex. C (ECF No. 43-1) ("Second Collins Rep.").

Dr. Collins is a Professor of Internal Medicine and Microbiology and Immunology at the University of Michigan Medical School. CV, dated July 12, 2023, filed as Ex. B (ECF No. 34-21) ("Collins CV"). She received her medical degree and her Ph.D. from Johns Hopkins University School of Medicine. Collins CV at 1. She completed a residency in internal medicine at Brigham and Women's Hospital in Boston, MA, and a fellowship in infectious diseases at various hospitals in the Boston area. *Id.* She also completed a research fellowship at Harvard University and a postdoctoral fellowship in immunology at Massachusetts Institute of Technology. *Id.* Dr. Collins is board certified in infectious disease. First Collins Rep. at 1. Her clinical practice at University of Michigan has focused on inpatients with complicated infectious disease problems. *Id.* Dr. Collins also runs a research laboratory that studies molecular mechanisms of HIV persistence, and she has published over 70 papers. *Id.*

First Report

After summarizing her own credentials and Petitioner's medical history, Dr. Collins provided her core opinion: that a CMV infection was more likely the cause of Petitioner's GBS than the flu vaccine. First Collins Rep. at 1-5. She began with an overview of GBS's known causes – highlighting the extent to which a variety of infections are deemed likely etiologic explanations. For example, a *C. jejuni* bacterial infection is a well-accepted GBS cause; the excess risk of GBS following this infection is 60-fold, and 20% of all GBS cases are attributable to this pathogen. *Id.*

[8] Because this matter turns on Respondent's success in carrying the "factor unrelated" burden, I begin my analysis with a summary of the opinion offered by Respondent's expert.

at 5; C. Tam et al., *Guillain-Barre Syndrome and Preceding Infection with Campylobacter, Influenza and Epstein-Barr Virus in the General Practice Research Database*, 2 PLOS One 1, 1 (2007), filed as Ex. A-3 (ECF No. 34-4). The risk of developing GBS following a *C. jejuni* infection is much higher than the background rate for individuals not so infected. First Collins Rep. at 5; N. Yuki, *Infectious Origins of, and Molecular Mimicry in, Guillain-Barre and Fisher Syndromes*, 1 The Lancet 29, 31 (2001), filed as Ex. A-4 (ECF No. 34-5) ("Yuki").

The same, evidentiarily-supported association exists for a CMV infection, and Dr. Collins provided a number of evidentiary bases for this aspect of her opinion. *See generally* First Collins Rep. at 5-6. A meaningful number of patients with GBS (up to a fifth) have been seen to possess IgM antibodies to CMV, establishing evidence of a recent infection – and the incidence of GBS after such a primary infection is comparable to the risk from a *C. jejuni* infection (compared to the background rate for the general population). *See* J.B. Winer et al., *A Prospective Study of Acute Idiopathic Neuropathy*, 51 J. of Neurol., Neurosurg. and Psych. 613, 616 (1998), filed as Ex. A-5 (ECF No. 34-6) ("Winer"); B.C. Jacobs et al., *The Spectrum of Antecedent Infections in Guillain-Barre Syndrome*, 51 Neurology 1110, 1113-14 (1998), filed as Ex. A-6 (ECF No. 34-7) ("Jacobs"); D. Orlikowski et al., *Guillain-Barre Syndrome following Primary Cytomegalovirus Infection: A Prospective Cohort Study*, 52 Clin. Infect. Diseases 837, 842 (2011), filed as Ex. A-8 (ECF No. 34-9) ("Orlikowski").

In describing the risk of GBS attributable to a CMV infection, Dr. Collins stressed several points specific to that virus. In particular, she noted that because the symptoms of a CMV infection are "usually self-limiting and not unique," they are not usually diagnosed at the time of infection. First Collins Rep. at 5. In fact, a CMV infection can manifest asymptomatically – with only some kinds of tests confirming its existence. *Id.* And (as discussed below), Dr. Collins felt Petitioner's own testing results were consistent in this regard. Dr. Collins also noted that medical science suggests that a CMV-caused GBS would be "more commonly associated with objective sensory defects," suggesting in turn "that the virus itself is directly or indirectly implicated and raises the question of whether CMV therapy would aid treatment for CMV-associated GBS." *Id.* at 6; Orlikowski at 843.

Dr. Collins also proposed that CMV-caused GBS might be mediated differently than other versions of GBS attributable to other kinds of triggers, maintaining that molecular mimicry (a common mechanistic explanation for how an autoimmune disease like GBS might occur)[9] might not be applicable in this context. First Collins Rep. at 6. Orlikowski, for example, proposed that the kind of anti-ganglioside antibodies often thought to be produced via molecular mimicry, and that might in turn attack nerve myelin cross-reactively, might be produced by a primary CMV

[9] "Molecular mimicry occurs as a result of amino acid sequences shared between a vaccine's viral or bacterial particles and structures in human tissue. . . The immune system's reaction to the foreign vaccine antigens produces immune cells that in turn can cross-react against the similar human tissue antigens, mistakenly identifying self-structures as foreign." *Mason v. Sec'y of Health & Hum. Servs.*, No. 17-1383V, 2022 WL 600415, at *5 (Fed. Cl. Spec. Mstr. Feb. 4, 2022).

infection, but were not also seen "to significantly influence the pathogenesis of CMV- GBS." First Collins Rep. at 6; Orlikowski at 843 ("Our results confirm that the anti-GM2 IgM response is more closely associated with primary CMV infection than with GBS . . . .").

Other studies emphasized different targets for autoimmune attack in GBS attributable to CMV infection, and were unable to identify evidence of CMV antigens as triggering the production of attacking autoantibodies. *See* S. Sawai et al., *Moesin is a Possible Target for Cytomegalovirus-related Guilllain-Barre Syndrome*, 83 Neurology 113, 116 (2014), filed as Ex. A-9 (ECF No. 34-10). Still more items of literature (including case series reports) seemed to associate a T cell-driven autoimmune attack in CMV-caused GBS, in comparison to patients whose GBS was associated with *C. jejuni*, or even no infection at all. R.D. Hadden et al., *Preceding Infectious, Immune Factors, and Outcome Guillain-Barre Syndrome*, 56 Neurology 758, 763 (2001), filed as Ex. A-13 (ECF No. 34-14). Dr. Collins acknowledged, however, that research on this topic had not yet reliably established any of its hypotheticals. First Collins Rep. at 6.

Dr. Collins provided several substantive bases for her view that a CMV infection best explained Petitioner's GBS. The medical record, she argued, established that Petitioner had experienced an active CMV infection around the time his GBS symptoms manifested. First Collins Rep. at 6. In support, she noted that Petitioner had tested positive for both IgG and IgM CMV antibodies when hospitalized in October 2020. *Id.* at 4. In addition, liver function tests revealed fluctuating abnormalities between mid- and late-October, when he was discharged. *Id.* at 3-4. Such a finding was associated with a CMV infection. *See* T. Friel, *Epidemiology, Clinical Manifestations, and Treatment of Cytomegalovirus Infection in Immunocompetent Adults*, Wolters Kluwer Health – UptoDate, https://www.uptodate.com/contents/epidemiology-clinical-manifestations-and-treatment-of-cytomegalovirus-infection-in-immunocompetent-adults (last updated Mar. 9, 2022), filed as Ex. A-1 (ECF No. 34-2) ("Friel"). Dr. Collins also noted that several test results from his October hospitalization showed an increase in absolute lymphocytes.[10] *Id.* Treaters had considered this evidence of "possible viral infection," and it was further indirect proof of the existence of the CMV infection. Ex. 2 at 30; Friel at 7.

The flu vaccine, by contrast, was less likely causal of GBS (even though the Vaccine Program clearly recognizes the claim). First Collins Rep. at 6-9. One 2001 UK study found less of a risk of developing GBS from the flu vaccine than from a wild flu infection. J. Stowe et al., *Investigation of the Temporal Association of Guillain-Barre Syndrome With Influenza Vaccine and Influenzalike Illness Using the United Kingdom General Practice Research Database*, 169 Am. J. of Epidemiology 382, 385 (2009), filed as Ex. D (ECF No. 52-1) ("Stowe"). Another epidemiologic prospective study reached a comparable conclusion. L. Grimaldi-Bensouda, *Guillain-Barre Syndrome, Influenzalike Illnesses, and Influenza Infection During Seasons With*

[10] A lymphocyte is type of white blood cell that determines the specificity of the body's immune response to infectious microorganisms and other foreign substances. *Lymphocyte*, Britannica Online, https://www.britannica.com/science/lymphocyte (last visited Mar. 17, 2025).

and Without Circulating A/H1N1 Viruses, Am. J. of Epidemiology 326, 334 (2011), filed as Ex. A-14 (ECF No. 34-15) ("Grimaldi-Bensuda") ("Influenza infections and influenza-like illnesses are likely risk factors for GBS and should therefore be considered serious confounders in future studies of GBS and vaccination. On the other hand, no new concerns about influenza vaccination, notably use of the A/H1N1 vaccine, have been identified"). A third article concluded the same, but also observed that some of the association between vaccination and GBS might be attributable to the fact that vaccination efforts also occurred in the midst of wild viral outbreaks, confounding findings that the vaccine's antigens alone were causal of GBS. S. Greene, *Guillain-Barre Syndrome, Influenza Vaccination, and Antecedent Respiratory and Gastrointestinal Infections: A Case Centered-Analysis in the Vaccine Safety Datalink 2009-2011*, 8 PLOS One 1, 6 (2013), filed as Ex. A-15 (ECF No. 34-16) ("Greene"). And another item of literature provided evidence that (given the undeniable connection between the wild flu virus and GBS) vaccination against the virus could be seen as *reducing* instances of GBS. C. Vellozzi et al., *Cumulative Risk of Guillain-Barre Syndrome Among Vaccinated and Unvaccinated Populations During the 2009 H1N1 Influenza Pandemic*, 104 Am. J. of Pub. Health 696, 700 (2014), filed as Ex. A-16 (ECF No. 34-17).

Second Report

Dr. Collins's supplemental report endeavored to respond to the arguments of Dr. Kelesidis (Petitioner's expert). Before entering into her point-by-point rebuttal, however, she noted the areas of agreement between both sides. In particular (and in addition to the fact that the GBS diagnosis is not disputed), she observed that Dr. Kelesidis had acknowledged (a) that most cases of GBS have an infectious etiology, (b) that Petitioner was found to have a CMV infection in the course of his hospitalization, and (c) that a CMV infection is a GBS risk factor. Second Collins Rep. at 1. Otherwise, Dr. Collins maintained that her opinion had withstood Petitioner's attacks.

First, she disputed Dr. Kelesidis's overall point that only a severe CMV infection with clear symptoms (of the kind that Petitioner did not report or experience) had the capacity to result in the "post-infectious etiology of his neurological manifestation." Second Collins Rep. at 1. The evidence offered by Dr. Kelesidis for this contention either focused on how GBS might present in severe cases, or discussed liver disturbances in the context of GBS, but without also reviewing the CMV-GBS association Dr. Collins maintained was established by independent literature. *See* A. de Jager, *Clinical Signs in Severe Guillain-Barre Syndrome: Analysis of 63 Patients*, 104 J. of Neuro. Sci. 143, 143 (1991), filed as Ex. 19 (ECF No. 42-3) ("de Jager"); P. Oomes et al., *Liver Function Disturbances in Guillain-Barre Syndrome: A Prospective Longitudinal Study in 100 Patients*, 46 Neurology J. 96, 98 (1996), filed as Ex. 20 (ECF No. 42-4). Nor was the presence of organ involvement in the context of a CMV infection a necessary prerequisite. Not only was there *some* evidence of possible organ involvement here (the liver function test results), but no independent evidence otherwise supported this argument. Second Collins Rep. at 3-4.

In fact, Dr. Collins maintained, evidence she had offered rebutted the contention that a CMV infection would *need* to be severe for GBS to be an associated risk. Orlikowski, for example, based its findings of a CMV-GBS relationship on individuals like Petitioner, where serum testing simply revealed evidence of IgM antibodies to CMV. Orlikowski at 839. This evidence of the presence of infection was accepted by the medical community, and thus Dr. Collins did not put stock in Dr. Kelesidis's contentions that "CMV IgM may not be specific for primary infection." Second Collins Rep. at 5. (She admitted, however, that in this case, unlike in Orlikowski, there was no evidence of the "avidity"[11] of Petitioner's IgG response, although IgG testing was consistent with a CMV infection's presence. Second Collins Rep. at 2.)

There was also "clear evidence" of the CMV virus circulating in Petitioner's blood. Second Collins Rep. at 2; Ex. 4 at 49, 23, 123. And Dr. Collins denied that "the amount of CMV DNA" found in testing was relevant to the severity of the infection. Second Collins Rep. at 5. The fact that GBS was post-infectious, she noted, was what was relevant (meaning that it could occur in the wake of a prior infection, regardless of severity). Nor did Dr. Collins consider significant the amount of CMV detected in cerebrospinal fluid testing, since in the majority of cases (as reflected in literature referenced by Dr. Kelesidis) "most (69%) of patients with CMV seropositivity and GBS did not have CMV DNA in their spinal fluid." *Id.*; C. Steininger, *Presence of Cytomegalovirus in Cerebrospinal Fluid of Patients with Guillain-Barre Syndrome*, 189 J. of Infectious Diseases 984, 986 (2004), filed as Ex. 33 (ECF No. 42-17) ("Steininger I").

In addition, Dr. Collins reiterated that CMV infections were more often than not asymptomatic in presentation. Second Collins Rep. at 3; Orlikowski at 839 (noting that a minority of studied patients presented with GI symptoms or fever). At the same time, however, Petitioner did complain of sensory deficits consistent with "CMV-associated GBS." Orlikowski at 840. And his liver function testing was also akin to what CMV-caused GBS would look like. Second Collins Rep. at 3.

Second, Dr. Collins took issue with Dr. Kelesidis's proposal that Petitioner's CMV infection was likely only a reactivation (and hence less likely to be causal of GBS). Second Collins Rep. at 4-5. She noted in response that the lab findings in this case confirming the presence of a CMV infection would have been deemed a sufficient basis to have included Petitioner in studies like Orlikowski, which considered those findings evidence of a "primary cytomegalovirus infection." Orlikowski at 837 ("We diagnosed 63 (12.4%) CMV-GBS cases by immunoglobulin (Ig) M detection and IgG avidity"). The fact that Petitioner was being treated for GBS was also not the cause of the alleged CMV reactivation. Rather, the infection likely predated findings of its

[11] IgG avidity is defined as "the strength with which IgG binds to antigenic epitopes expressed by a given protein. . . Low CMV IgG avidity is an accurate indicator of primary infection within the preceding 3 to 4 months, whereas high avidity excludes primary infection within the preceding 3 months." H. Prince & M. Lape-Nixon, *Role of Cytomegalovirus (CMV) IgG Avidity Testing in Diagnosing Primary CMV Infection During Pregnancy*, 21 Clin. and Vaccine Immunology 1377, 1377 (2014), filed as Ex. 61 (ECF No. 49-24) ("Prince"). IgG avidity testing "is increasingly considered the 'gold standard' for distinguishing primary from nonprimary CMV infection." Prince at 1377.

presence, since "[i]t takes time following exposure to a virus for IgM and IgG to become detectable." Second Collins Rep. at 6. Existing studies supported the conclusion that Petitioner's infection exposure began several days before testing revealed it. *Id.*; Ex. 4 at 126. But Petitioner's GBS symptoms did not manifest until October 15th (although the record of this visit suggests an onset five days prior). And treatments that might have had an impact on CMV reactivation, like steroids, began later as well. Dr. Collins did not deem Petitioner's age to make it more likely any CMV infection he experienced was a reactivation. Second Collins Rep. at 6.

More significantly, however, Dr. Collins noted a lack of evidence offered by Petitioner in support of the conclusion that only a primary infection *could* present a GBS risk. Second Collins Rep. at 5, 7-8. Dr. Kelesidis seemed to assume only immunocompromised individuals would be threatened by a reactivated infection, but offered no good evidence to support this supposition. *Id.* at 7. No other evidence offered by Petitioner, Dr. Collins argued, established a lack of association between GBS and reactivation of CMV – while literature suggested that reactivation *could* still be GBS-associated. Second Collins Rep. at 6; C. Steininger, *Primary Cytomegalovirus Infection in Patients with Guillain-Barre Syndrome*, 183 J. of Neuroimmunology 214, 218 (2007), filed as Ex. 32 (ECF No. 42-16) ("Steininger II"). The degree of risk had not been shown to be dependent on the proper characterization or nature of the infection.

Third, Dr. Collins reiterated her view that the lab findings she had highlighted (such as lymphocytosis or liver function/transaminitis) were consistent with a CMV infection. Second Collins Rep. at 4. Dr. Kelesidis had suggested that the timing of different findings during the course of Petitioner's treatment was evidence of an "inconsistent progression," especially in light of the lack of confirming symptoms manifestations. *Id.* But the core fact of Petitioner's infection (as evidenced by antibody findings) was not impacted by these other lab findings (even though Dr. Collins had deemed them significant). And no evidence referenced by Dr. Kelesidis stood for the proposition that the progression of these other lab findings had anything to do with the degree of GBS risk in the wake of a CMV infection. *Id.*

Dr. Collins went on to discuss what the record indicated about Petitioner's treaters, and their possible views about the etiology of his GBS and/or relationship to a prior infection. Second Collins Rep. at 7. Although the record does show that some infectious consult occurred in the course of Petitioner's treatment, Dr. Collins noted that it has an informal and cursory nature, referred to in the actual record as "curbside" (meaning casual and based on a very brief summary). *Id.* The intent of this evaluation, moreover, was only to decide if the discovered CMV infection warranted treatment – a consideration independent of whether the infection was associated with Petitioner's GBS (something that this set of records said nothing about). Dr. Collins concurred with the decision not to treat, observing that "CMV typically resolves without specific treatment in an immunocompetent person." *Id.*

Dr. Collins concluded her second report with more discussion of the relative risks of GBS post-vaccination versus post-infection and engaged in a pointed effort to rebut some of Dr.

Kelesidis's contentions on this topic. Second Collins Rep. at 8-11. However, I do not deem that question to be particularly in dispute – and since this is otherwise a Table claim, the causal association of the flu vaccine with GBS is also not in question. Accordingly, the debate between the experts on this point is academic for present purposes.

B. *Petitioner's Expert – Theodoros Kelesidis, M.D., Msc., Ph.D.*

Dr. Kelesidis is (like Dr. Collins) an infectious disease specialist and medical academic, and he authored two reports in support of Petitioner. Report, dated Jan. 11, 2024, filed as Ex. 17 (ECF No. 42-1) ("First Kelesidis Rep."); Report, dated Aug. 2, 2024, filed as Ex. 39 (ECF No. 49-2) ("Second Kelesidis Rep.").

Dr. Kelesidis is an Associate Professor in the Division of Infectious Diseases at University of Texas Southwestern Medical Center. CV, dated Jan. 11, 2024, filed as Ex. 18 (ECF No. 42-2) ("Kelesidis CV"). He received his medical degree from Athens University Medical School in Athens, Greece, and received his Ph.D. from the Department of Microbiology, Immunology and Molecular Genetics at UCLA. Kelesidis CV at 1. Dr. Kelesidis completed a residency in internal medicine at St. Elizabeth's Medical Center in Boston, MA and completed a fellowship in infectious diseases at UCLA. *Id.* He is board-certified in infectious disease. First Kelesidis Rep. at 1. His clinical practice has focused on patients with complicated infectious disease problems and his research laboratory studies molecular mechanisms of immunopathogenesis of viral infections. *Id.* Dr. Kelesidis has published over 150 papers. *Id.*

First Report

Dr. Kelesidis accepted both Dr. Collins's summation of the medical history, as well as Petitioner's ultimate GBS diagnosis. First Kelesidis Rep. at 1. But he denied a CMV infection could explain Petitioner's GBS, and he provided several points to support his opinion.

Dr. Kelesidis disputed that the medical record established that Petitioner had experienced any infection of sufficient severity to cause GBS. First Kelesidis Rep. at 1-3. Most cases of GBS deemed viral in origin would be preceded by evidence of infectious-associated symptoms, from upper respiratory complaints to gastrointestinal issues. *Id.* at 1; de Jager at 144. But the medical record in this case revealed no such complaints prior to Petitioner's onset of neurologic symptoms. He had not even experienced a sore throat (a common CMV-associated symptom), nor had he been caring for an infant or young child – the "main vector in primary CMV infections in late adulthood." First Kelesidis Rep. at 2; A. Al-Omari et al., *Cytomegalovirus Infection in Immunocompetent Critically Ill Adults: Literature Review*, 6 Annals of Intensive Care 1, 2 (2016), filed as Ex. 22 (ECF No. 42-6) ("Al-Omari"). (The record in this case does establish Petitioner had an infant daughter, although there is no evidence the child displayed any infection-related symptoms before Petitioner's onset. Ex. 13 at 2. And Al-Omari says only that CMV is more commonly acquired in childhood – not that children transmit it to adults).

CMV, Dr. Kelesidis agreed, could be largely subclinical in its course, and "[m]ost healthy individuals who acquire CMV infection are able to clear the infection within a short length of time with no adverse sequelae." First Kelesidis Rep. at 2. But severe symptoms were also possible, and they could run the gamut, from GI-associated concerns to "life-threatening complications" that could result in organ damage. *Id.* Liver impact, in Dr. Kelesidis's view, would be rare, and if present, would result in obvious secondary symptoms, "including fever and mononucleosis like syndrome (sore throat, swollen lymph nodes)." *Id.*

Dr. Kelesidis proposed that the record evidence only established, at most, a reactivation of a prior CMV infection – insufficient in his view to be causal of GBS. *See generally* First Kelesidis Rep. at 3-4. He agreed that Petitioner as of October 15, 2020, displayed elevated liver enzymes, or "transaminitis," but maintained that the levels subsequently "self-resolved." *Id.* at 2. The same was true for test results demonstrating lymphocytosis, with Dr. Kelesidis considering the results to show only mild elevations that could not be considered atypical – and which also did not persist. *Id.* It certainly was not the degree of abnormality which had been seen in studies of CMV infections involving mononucleosis. D. Felsenstein et al., *Phenotypic Properties of Atypical Lymphocytes in Cytomegalovirus-Induced Mononucleosis*, 152 J. of Infect. Diseases 198, 201-02 (1985), filed as Ex. 27 (ECF No. 42-11). And lymphocytosis was not specific to a CMV infection in any event, further diminishing the significance of those findings here. First Kelesidis Rep. at 2. Otherwise, Petitioner had displayed no signs of an active CMV infection. For example, he had no fever, and no signs of the kind of mononucleosis (associated with CMV) that might establish the presence of an infection. *Id.*

A CMV reactivation, was, in Dr. Kelesidis's view, more likely to have occurred in this case. First Kelesidis Rep. at 3. As a general matter, an adult of Petitioner's age would be unlikely to have never had CMV in his life before – and CMV is known to be a virus that could remain latent in the body, well after exposure to it as a child. Friel at 2; P. Lachance, *Association Between Cytomegalovirus Reactivation and Clinical Outcomes in Immunocompetent Critically Ill Patients: A Systematic Review and Meta-Analysis*, 4 Open Forum Infect. Diseases 1, 1 (2017), filed as Ex. 29 (ECF No. 42-13) ("Lachance"). The testing performed on Petitioner also revealed an overall low viral load – especially in comparison with the kind of especially-virulent infection that would be associated with significant pathology. P. Griffiths & M. Reeves, *Pathogenesis of Human Cytomegalovirus in the Immunocompromised Host*, 19 Nature Rev. Microbio. 759, 759 (2021), filed as Ex. 31 (ECF No. 42-15) (". . . [I]f the immune system is compromised, HCMV can replicate to high levels and cause serious end organ disease"). The presence of IgM CMV antibodies, even in patients with GBS, was not evidence of an active infection, as recognized in relevant literature. Steininger II at 218. Petitioner's cerebrospinal fluid testing had not revealed evidence of an active infection (as it should have if his GBS were CMV-caused). *Id.* at 215. In addition, CMV reactivation could have occurred *due* to Petitioner's GBS (as opposed to the latter caused by the former). *See* Lachance at 1 ("This state of latency allows CMV to reactivate when host defenses become compromised, such as in critical illness. Cytomegalovirus reactivation in

critically ill patients is well recognized with as high as 71% incidence. . ."). In fact, Petitioner's treaters had expressed the view that Petitioner's CMV infection was merely a reactivation "in the setting of illness," and that it did not merit treatment on its own. First Kelesidis Rep. at 3.

A reactivated CMV infection, moreover, was less likely to result in GBS. First Kelesidis Rep. at 3-4. But to establish this point, Dr. Kelesidis relied primarily on the *absence* of affirmative evidence from literature that reactivated CMV infections in "immunocompetent patients" (which would presumably be a fair characterization of Petitioner) were associated with GBS. At most, some evidence (such as case reports) suggested that individuals severely ill from a CMV infection might in rare circumstances secondarily develop GBS. *Id.* at 4.

Dr. Kelesidis felt, by contrast, that it was far more likely Petitioner's GBS was vaccine-caused. First Kelesidis Rep. at 4-7. In so opining, he summarized existing medical science supporting the mechanism for an association (occurring via molecular mimicry between vaccine antigens and self-structures on the nerve myelin) and highlighted a number of studies that he felt continued to show a possible link from an epidemiologic standpoint (in contrast to the items of literature offered by Dr. Collins to undermine that conclusion). *Id.* at 5-6.[12] And he deemed contrary evidence to be limited either by methodologic issues (such as insufficiently-large samples) or by the fact that rare occurrences like GBS cannot be predicted with epidemiology. *Id.* at 6. Dr. Kelesidis also proposed that an onset of a week to two weeks post-vaccination was medically acceptable, based on the likely autoimmune mechanism at issue – molecular mimicry. *Id.* at 4.

Second Report

Dr. Kelesidis's final report attempted to defend his overall opinion that Petitioner's CMV infection could not have caused his GBS.[13] He sought to bulwark his argument that Petitioner's infection was overall insufficiently severe to have resulted in GBS. Second Kelesidis Rep. at 1-4. He again emphasized the lack of evidence of any significant clinical manifestations of a CMV infection prior to its discovery. *Id.* at 1. He also maintained that the absence of such symptoms *plus* results from "IgG avidity testing," did not support the conclusion that Petitioner had

[12] I do not include a specific discussion of these items of literature – both because the vaccine's capacity to cause GBS is not relevant in the context of a Table claim (where causation is presumed), but also because Dr. Collins's efforts were focused more on an equally-indisputable point: that the wild flu virus is *far more* likely to cause GBS than the flu vaccine. And a distinction between population-level risk and individual risk matters not. Epidemiologic evidence on this sub-issue (vaccination risk versus wild virus risk) still preponderates against the relative risk of vaccination compared to infection. Arguments that "no study can ever disprove the possibility of individual causation" misapprehend the weight that such evidence deserves – *when it exists*. *Kelly v. Sec'y of Health & Hum. Servs.*, No. 16-878V, 2021 WL 5276373, at *26 n.24 (Fed. Cl. Spec Mstr. Oct. 18, 2021).

[13] Dr. Kelesidis also repeated many of his arguments about the flu vaccine's association with GBS (*see generally* Second Kelesidis Rep. at 11-12), but I do not recapitulate these secondary points, for the same reasons I have not addressed their treatment in Dr. Collins's second report.

experienced a primary infection. *Id.* at 1-2, *citing* Steininger II at 217-18. And Dr. Collins, as noted above, had admitted that these findings were not available.

This left Dr. Collins to rely on secondary proof, like the liver functionality testing results. But Dr. Kelesidis deemed these nonspecific in nature, and therefore likely evidence of other kinds of issues beyond a CMV infection. Second Kelesidis Rep. at 2. In fact, the liver function results could have been the product of Petitioner's self-medication with Tylenol to treat his headaches before he sought medical assistance. *Id.*; Jorge Herrera & Alisa Likhitsup, *Medications and the Liver*, Am. College of Gastroenterology, https://gi.org/topics/medications-and-the-liver (last updated August 2022), filed as Ex. 60 (ECF No. 49-23) (stating that Tylenol is the best-known medication that can cause damage to the liver).

The record in fact, Dr. Kelesidis maintained, was consistent with the conclusion that Petitioner had experienced a reactivated infection rather than primary. Second Kelesidis Rep. at 5-8. Because of a lack of IgG avidity testing, Orlikowski could not be credibly invoked by Dr. Collins as supporting the conclusion that Petitioner's infection was primary. *Id.* at 6. Other evidence offered by Dr. Collins stood as support for CMV reactivation being a risk factor for GBS *only* in cases where the individual was "critically ill," and thus likely immunocompromised. *Id.* at 6-7; A. Caliendo, *Approach to the Diagnosis of Cytomegalovirus Infection*, Wolters Kluwer Health – UptoDate, https://www.uptodate.com/contents/approach-to-the-diagnosis-of-cytomegalovirus-infection (last updated May 16, 2024), filed as Ex. A-7 (ECF No. 34-8); L. Papazian et al., *Cytomegalovirus Reactivation in ICU Patients*, 42 Intensive Care Med. 28, 28-29 (2016), filed as Ex. 49 (ECF No. 49-12). The IgM evidence was not necessarily dispositive proof of a recent infection, since IgM antibodies could persist up to a year after initial encounter with the infectious agent or could simply reflect reactivation. Second Kelesidis Rep. at 7; H. Prince & M. Lape-Nixon, *Role of Cytomegalovirus (CMV) IgG Avidity Testing in Diagnosing Primary CMV Infection During Pregnancy*, 21 Clin. and Vaccine Immunology 1377, 1378 (2014), filed as Ex. 61 (ECF No. 49-24) ("Prince"). Petitioner's adult status made it likely he had been exposed to CMV in his life before, and because the CMV virus tended to remain latent in the body, it was simply more credible that this is what had occurred in this case. Second Kelesidis Rep. at 11.

Such a less-virulent reactivated infection was unlikely to result in GBS. Second Kelesidis Rep. at 7. In so arguing, Dr. Kelesidis maintained that no mechanism had been identified linking *any* form of CMV infection with GBS, proposing that the studies Dr. Collins generally relied upon all displayed "major limitations" that made it impossible to give them probative weight. *Id.* at 7-8. Otherwise, Dr. Kelesidis maintained that Petitioner's own testing levels for CMV viral load were low, consistent with a reactivation, and that it was more likely his GBS contributed to that reactivation than vice-versa. *Id.* at 8.

Dr. Kelesidis further sought to identify record proof that he deemed more consistent with a vaccine-caused GBS than infectious-caused. Had Petitioner's GBS been due to a CMV infection, progression of the illness process should have been evident in testing. Second Kelesidis Rep. at 4.

But the "inconsistent presence of lymphocytosis and atypical lymphocytes" seen in testing results in this case did not make an infectious cause more likely, and were in fact nonspecific, or even due to GBS. *Id.* And Petitioner's treaters did not deem the CMV infection to be a possible causal factor, with Dr. Kelesidis downplaying the "curbside" nature of the infectious consult Petitioner received. *Id.* at 8.

III. Procedural History

This claim was initiated in March 2022. In April 2023, Respondent filed his Rule 4(c) Report contesting entitlement. Respondent filed an expert report from Dr. Collins in July 2023, and Petitioner filed a responsive report from Dr. Kelesidis in January 2024. Three months later, in April 2024, Respondent filed a supplemental report from Dr. Collins. In August 2024, Petitioner filed his final expert report from Dr. Kelesidis, along with a Motion for Ruling on the Record. Respondent filed his Opposition and Petitioner followed up with his Reply. The matter is now ripe for resolution.

IV. Parties' Arguments

Respondent

Respondent contends that Petitioner's GBS was caused by an active, if asymptomatic, CMV infection, as supported by definitive laboratory testing results. Opp. at 1, 8-9. According to Friel, "acute [CMV] infection is strongly suggested by the detection of either CMV-specific IgM antibody or a four-fold or greater rise in CMV-specific IgG performed in paired specimens obtained at least two weeks apart." Friel at 5. And Petitioner possessed CMV-specific IgM antibodies. Opp. at 9; Ex. 4 at 126. While Petitioner argues that he needed to have an IgG avidity score performed simultaneously with his IgM result to establish that he had a recent primary CMV infection, IgM results without avidity scores were used in multiple peer-reviewed articles evaluating the associations between CMV and GBS. Opp. at 10; Second Collins Rep. at 5. Because Petitioner had positive IgM, he would have been included in studies showing that a recent CMV infection is a risk factor for the development of GBS. Opp. at 12; *see also* Winer at 2 ("The presence of IgM antibodies was taken to indicate recent CMV infection"); Jacobs at 2 (CMV infection was "defined as the presence of IgM"). Moreover, Petitioner argues that the lack of another test is inadequate to overcome Respondent's preponderant showing. Opp. at 10.

Additionally, Petitioner had elevated atypical lymphocytes at ten percent. Opp. at 9; Ex. 4 at 123. According to the medical literature, "the presence of more than [ten] percent atypical lymphocytes on peripheral blood smear" is a hematologic abnormality that indicates an active CMV infection. Friel at 6-7. Further, Petitioner had abnormal AST and ALT levels, and "[s]ubclinical transaminitis is the most common finding in immunocompetent patients[.]" Opp. at 9; Friel at 9. Petitioner also had elevated liver transaminases. Opp. at 9; Ex. 4 at 123. All of this led Dr. Collins to conclude that Petitioner likely had an ongoing CMV infection at the time of vaccination. Opp. at 10.

Respondent further argues that Petitioner not only failed to establish that his CMV infection was a reactivation rather than primary infection, but that this distinction matters. Opp. at 10. In support of his argument, Petitioner notes that he did not have any symptoms. *Id.* But Respondent contends that this is not unusual, since "[i]nfection in the immunocompetent host is generally asymptomatic[.]" *Id.* at 9 (citing Friel at 1).

Petitioner also argues that his CMV viral load was low, but as Dr. Collins explains, CMV viral load is related to severity, not whether an infection is present in the first place. Opp. at 11. Petitioner's claim that GBS or steroid use can reactivate a CMV infection is simply unsupported. *Id.* Respondent notes that Petitioner's only evidence in support of this claim is LaChance, which discusses CMV activation in critically ill patients, unlike Petitioner. *Id.*; LaChance at 1. Opp. at 11. Finally, the infectious disease team's cursory statement that Petitioner's CMV was "likely re-activation [in setting of] recent illness, would not treat" does not overcome Respondent's showing. *Id.* Curb-siding reflects a minimal clinical evaluation, and CMV typically resolves without specific treatment in an immunocompetent person. *Id.*

Respondent also argues that Petitioner has not provided any support for his contention that severe or symptomatic CMV infection is a prerequisite to general causation of GBS. Opp. at 13. Dr. Kelesidis recognizes that most primary CMV infections are subclinical, and that "active infection has been linked to GBS." *Id.*; Second Collins Rep. at 3-4; First Kelesidis Rep. at 4. And Dr. Kelesidis's claim that viral damage and release of antigens increases based on the severity of the infection stands in stark contrast to his subsequent attempt to ascribe causation to the flu vaccine, which did not cause Petitioner to experience effects anywhere near the same level as a viral infection. Opp. at 13.

Respondent maintains that a great deal of data supports the conclusion that infection is an important cause of GBS. Opp. at 14; *see also* Yuki at 1; Greene at 1-8. Notably, CMV "is the most common viral antecedent infection" of GBS. Opp. at 14 (citing Yuki at 1). Respondent argues, contrary to Petitioner's demand, that he need not elucidate a mechanism causally connecting CMV to GBS. *Id.* at 15. Respondent's theory is sufficiently supported by expert testimony and epidemiological evidence. *Id.* Moreover, some Program decisions support Respondent's contentions about the greater chance of a wild flu virus causing GBS over the flu vaccine. *Bielak v. Sec'y of Health & Hum. Servs.*, No. 18-761V, 2023 WL 35509, at *30-31 (Fed. Cl. Spec. Mstr. Jan. 3, 2023) (noting "viral infections have also been found associated with GBS—further supporting the contention that the flu vaccine could be causal"); *Mason v. Sec'y of Health & Hum. Servs.*, No. 17-1383V, 2022 WL 600415, at *5-6, *26 (Fed. Cl. Spec. Mstr. Feb. 4, 2022) (summarizing petitioner's expert's molecular mimicry theory, which hinged upon research associating infections, including CMV, with GBS).

Respondent concludes with the proposition that Petitioner's CMV infection caused his GBS within a medically acceptable timeframe. Opp. at 16. Dr. Collins opined that Petitioner's CMV "infection was recent and ongoing because the IgM was positive and because viral DNA

was detected." First Collins Rep. at 6. Even if Petitioner had a CMV reactivation, it is the infection *per se* that is significantly associated with GBS. Opp. at 17; Second Collins Rep. at 4-8. In contrast to the strong evidence linking CMV infections and GBS, the medical literature does not show an association between the flu vaccine and GBS. Opp. at 18; Stowe at 1-2; Grimaldi-Bensuda at 1-2; Greene at 1. In sum, the record shows that the flu vaccine had no more than a coincidental relationship to Petitioner's GBS. Opp. at 20; First Collins Rep. at 9. Petitioner's active CMV infection was the preponderant sole substantial cause of Petitioner's GBS. Opp. at 20.

Petitioner

Petitioner asserts he can meet the elements for a Table claim of GBS after receipt of the seasonal flu vaccine. Mot. at 18. Five days after receiving the vaccine on October 5, 2020, Petitioner developed tingling and generalized weakness in bilateral hands, feet, and mouth. *Id.*; Ex. 2 at 43. Two weeks later (on October 25, 2020), Petitioner was diagnosed with "GBS [ ] likely in the setting of recent flu vaccine" and was treated with a three-day course of IVIg. Mot. at 19; Ex. 4 at 48-49. Because Petitioner developed GBS within the statutorily-defined timeframe of 3-42 days, he has satisfied the *prima facie* criteria for an on-Table claim. Mot. at 19.

Respondent asserts that Petitioner's GBS was caused by an active CMV infection. Mot. at 19. But Petitioner denies the "factor unrelated" burden has been carried. Respondent has failed to demonstrate that Petitioner was experiencing an active CMV infection, or that such an infection was the sole substantial factor leading to his development of GBS. *Id.*

Petitioner argues that Respondent's expert, Dr. Collins, offered studies that failed to elucidate a mechanism linking CMV to the development of GBS. Mot. at 19-20. Orlikowski, for example, found that the serum GM2 IgM – while closely associated with primary CMV infection – is unlikely to significantly influence the development of CMV-associated GBS. *Id.* at 20 (citing Orlikowski at 843). Unable to prove a causal mechanism, Dr. Collins relies on small epidemiological studies that may establish an association, but do not establish causation. Mot. at 20. Winer and Jacobs both identified a small subset of patients with CMV-specific IgM antibodies, which are thought to be indicative of a recent primary CMV infection (although a latent CMV infection could not be excluded). *Id.* at 20-22; Winer at 617; Jacobs at 1110. The authors discussed possible mechanisms to explain the link between CMV and GBS, but Dr. Collins questioned the applicability of some of these mechanisms and explicitly rejected the molecular mimicry theory. Mot. at 21-22; First Collins Rep. at 6. Dr. Collins's reliance on Orlikowski is also inapplicable because the patients in Orlikowski were experiencing a "recent primary infection," while the medical records in this case support a finding that Petitioner was experiencing a *reactivation* of CMV secondary to his onset of GBS. Mot. at 23; Ex. 4 at 49, 93.

Respondent also points to Petitioner's abnormal LFTs, lab test results identifying IgM and IgG antibodies to CMV, the presence of lymphocytosis, and neurological manifestations as all "consistent with CMV-associated GBS." Mot. at 24-30. Dr. Collins argues that a person with an

asymptomatic CMV infection can exhibit mild increases in liver transaminases, without additional symptoms of infection. *Id.* at 25; First Collins Rep at 5-6. But Friel, which Dr. Collins uses to support her argument, indicates that increases in liver transaminases can be observed in *symptomatic* CMV infections. Mot. at 25; Friel at 9. Petitioner points out that his transient transaminitis was more likely caused by his GBS, NSAID medications, and/or his IVIg treatments. Mot. at 26.

Dr. Collins also relies on lab tests that show the presence of both IgM and IgG antibodies to CMV. Mot. at 27. But these findings do not allow for the differentiation between a recent or active primary infection, in comparison to CMV reactivation in the setting of a critical illness. *Id.* Petitioner explains that the presence of IgG antibodies indicates that a person was, at some point in their life, infected with CMV. *Id.* at 28. While IgM antibodies can indicate a recent primary infection, they are also produced during a secondary infection and reactivation. *Id.*; *see also* Steininger II at 215; Steininger I at 984. The only reliable means of distinguishing between an active CMV infection and a latent CMV infection is IgG avidity testing, which Petitioner did not undergo. Mot. at 29. Therefore, the available antibody studies in this case do not stand as strongly persuasive evidence of a primary CMV infection. *Id.* at 30.

Respondent also argues that Petitioner's lymphocytosis and elevated levels of atypical lymphocytes suggest the presence of an ongoing CMV infection, but he fails to acknowledge that both are observed in a number of conditions besides CMV infection. Mot. at 30; H. Hamad & A. Mangla, *Lymphocytosis*, StatPearls (2023), filed as Ex. 48 (ECF No. 49-11); T. Shiftan & J. Mendelsohn, *The Circulating "Atypical" Lymphocyte*, 9 Hum. Pathology 51 (1978), filed as Ex. 47 (ECF No. 49-10).

Finally, Respondent claims that Petitioner demonstrated neurological manifestations that are most consistent with CMV-associated GBS, like objective sensory defects. Mot. at 31. Petitioner responds by arguing that this opinion far exceeds Dr. Collins's area of expertise, given her lack of training in neurology. *Id.* at 32. Furthermore, Dr. Collins ignores the fact that the GBS subtype Petitioner was diagnosed with, AIDP, is typically characterized by symmetric motor flaccid weakness *and* sensory abnormalities. *Id.* at 33. And the literature that Dr. Collins uses to support her argument, Friel, describes inconsistent results in studies seeking to identify common neurologic manifestations that could help distinguish CMV-associated GBS. *Id.*; Friel at 10. Facial nerve palsies appear to be the only neurologic manifestation that has consistently been observed at higher rates in CMV-associated GBS, and Petitioner never demonstrated facial nerve palsy. Mot. at 33-34; Friel at 10.

Turning to the third *Althen* prong, Petitioner argues that Respondent failed to demonstrate a proximate temporal relationship between Petitioner's CMV and GBS. Mot. at 37. The studies offered by Respondent suggest that the onset of GBS typically occurs two weeks after a CMV infection. *Id.* at 34; Winer at 4; Orlikowski at 839. Respondent contends that Petitioner was infected with CMV prior to October 13, 2020, but this assumption is based on medical literature

describing IgG and IgM responses to coronavirus infections – not CMV. Mot. at 35; (citing H. Hou et al., *Detection of IgM and IgG Antibodies in Patients with Coronavirus Disease 2019*, 9 Clin. & Translational Immunology 1, 1-6 (2020), filed as Ex. C-2 (ECF No. 43-3); X. Liu et al., *Patterns of IgG and IgM Antibody Response in COVID-19 Patients*, 9 Emerging Microbes & Infections 1, 1-6 (2020), filed Ex. C-3 (ECF No. 43-4)). CMV-specific literature does not support Dr. Collins's proposed timeline of events, as IgM antibodies to CMV can persist for over a year. Mot. at 36; Prince at 1379. Therefore, the presence of IgM antibodies in Petitioner's blood sheds no light on when Petitioner contracted his CMV infection. Mot. at 37.

V. Applicable Legal Standards

A. *Petitioner's Overall Burden in Vaccine Program Cases*

To receive compensation in the Vaccine Program, a petitioner must prove either: (1) that he suffered a "Table Injury"—i.e., an injury falling within the Vaccine Injury Table—corresponding to one of the vaccinations in question within a statutorily prescribed period of time or, in the alternative, (2) that his illnesses were actually caused by a vaccine (a "Non-Table Injury"). *See* Sections 13(a)(1)(A), 11(c)(1), and 14(a), as amended by 42 C.F.R. § 100.3; § 11(c)(1)(C)(ii)(I); *see also Moberly*, 592 F.3d at 1321; *Capizzano v. Sec'y of Health & Hum. Servs.*, 440 F.3d 1317, 1320 (Fed. Cir. 2006).[14] Mr. Taylor asserts a Table claim of GBS after receipt of the flu vaccine.

For both Table and Non-Table claims, Vaccine Program petitioners bear a "preponderance of the evidence" burden of proof. Section 13(1)(a). That is, a petitioner must offer evidence that leads the "trier of fact to believe that the existence of a fact is more probable than its nonexistence before [he] may find in favor of the party who has the burden to persuade the judge of the fact's existence." *Moberly*, 592 F.3d at 1322 n.2; *see also Snowbank Enter. v. United States*, 6 Cl. Ct. 476, 486 (1984) (mere conjecture or speculation is insufficient under a preponderance standard). Proof of medical certainty is not required. *Bunting v. Sec'y of Health & Hum. Servs.*, 931 F.2d 867, 873 (Fed. Cir. 1991). In particular, a petitioner must demonstrate that the vaccine was "not only [the] but-for cause of the injury but also a substantial factor in bringing about the injury." *Moberly*, 592 F.3d at 1321 (quoting *Shyface*, 165 F.3d at 1352–53); *Pafford v. Sec'y of Health & Hum. Servs.*, 451 F.3d 1352, 1355 (Fed. Cir. 2006). A petitioner may not receive a Vaccine Program award based solely on his assertions; rather, the petition must be supported by either medical records or by the opinion of a competent physician. Section 13(a)(1).

In attempting to establish entitlement to a Vaccine Program award of compensation for a Non-Table claim, a petitioner must satisfy all three of the elements established by the Federal

[14] Decisions of special masters (some of which I reference in this ruling) constitute persuasive but not binding authority. *Hanlon v. Sec'y of Health & Hum. Servs.*, 40 Fed. Cl. 625, 630 (1998). By contrast, Federal Circuit rulings concerning legal issues are binding on special masters. *Guillory v. Sec'y of Health & Hum. Servs.*, 59 Fed. Cl. 121, 124 (2003), *aff'd* 104 F. Appx. 712 (Fed. Cir. 2004); *see also Spooner v. Sec'y of Health & Hum. Servs.*, No. 13-159V, 2014 WL 504728, at *7 n.12 (Fed. Cl. Spec. Mstr. Jan. 16, 2014).

Circuit in *Althen*, 418 F.3d at 1278: "(1) a medical theory causally connecting the vaccination and the injury; (2) a logical sequence of cause and effect showing that the vaccination was the reason for the injury; and (3) a showing of proximate temporal relationship between vaccination and injury."

Each of the *Althen* prongs requires a different showing. Under *Althen* prong one, petitioners must provide a "reputable medical theory," demonstrating that the vaccine received *can cause* the type of injury alleged. *Pafford*, 451 F.3d at 1355–56 (citations omitted). To satisfy this prong, a petitioner's theory must be based on a "sound and reliable medical or scientific explanation." *Knudsen v. Sec'y of Health & Hum. Servs.*, 35 F.3d 543, 548 (Fed. Cir. 1994). Such a theory must only be "legally probable, not medically or scientifically certain." *Id.* at 549.

Petitioners may satisfy the first *Althen* prong without resort to medical literature, epidemiological studies, demonstration of a specific mechanism, or a generally accepted medical theory. *Andreu*, 569 F.3d at 1378–79 (citing *Capizzano*, 440 F.3d at 1325–26). Special masters, despite their expertise, are not empowered by statute to conclusively resolve what are essentially thorny scientific and medical questions, and thus scientific evidence offered to establish *Althen* prong one is viewed "not through the lens of the laboratorian, but instead from the vantage point of the Vaccine Act's preponderant evidence standard." *Id.* at 1380. Accordingly, special masters must take care not to increase the burden placed on petitioners in offering a scientific theory linking vaccine to injury. *Contreras*, 121 Fed. Cl. at 245.

In discussing the evidentiary standard applicable to the first *Althen* prong, the Federal Circuit has consistently rejected the contention that it can be satisfied merely by establishing the proposed causal theory's scientific or medical *plausibility*. *See Boatmon v. Sec'y of Health & Hum. Servs.*, 941 F.3d 1351, 1359 (Fed. Cir. 2019); *see also LaLonde v. Sec'y of Health & Hum. Servs.*, 746 F.3d 1334, 1339 (Fed. Cir. 2014) ("[h]owever, in the past we have made clear that simply identifying a 'plausible' theory of causation is insufficient for a petitioner to meet her burden of proof" (citing *Moberly*, 592 F.3d at 1322)); *Howard v. Sec'y of Health & Hum. Servs.*, No. 16-1592V, slip op. at *6 (Fed. Cl. Feb. 27, 2023) (confirming that "[t]he standard has been preponderance for nearly four decades"), *appeal docketed*, No. 2023-1816 (Fed. Cir. Apr. 28 2023). Otherwise, petitioners *always* have the ultimate burden of establishing their Vaccine Act claim with preponderant evidence. *W.C. v. Sec'y of Health & Hum. Servs.*, 704 F.3d 1352, 1356 (Fed. Cir. 2013) (citations omitted); *Tarsell v. United States*, 133 Fed. Cl. 782, 793 (2017) (noting that *Moberly* "addresses the petitioner's overall burden of proving causation-in-fact under the Vaccine Act" by a preponderance standard).

The second *Althen* prong requires proof of a logical sequence of cause and effect, usually supported by facts derived from a petitioner's medical records. *Althen*, 418 F.3d at 1278; *Andreu*, 569 F.3d at 1375–77; *Capizzano*, 440 F.3d at 1326; *Grant v. Sec'y of Health & Hum. Servs.*, 956 F.2d 1144, 1148 (Fed. Cir. 1992). In establishing that a vaccine "did cause" injury, the opinions and views of the injured party's treating physicians are entitled to some weight. *Andreu*, 569 F.3d

at 1367; *Capizzano*, 440 F.3d at 1326 ("medical records and medical opinion testimony are favored in vaccine cases, as treating physicians are likely to be in the best position to determine whether a 'logical sequence of cause and effect show[s] that the vaccination was the reason for the injury'") (quoting *Althen*, 418 F.3d at 1280). Medical records are generally viewed as particularly trustworthy evidence, since they are created contemporaneously with the treatment of the patient. *Cucuras v. Sec'y of Health & Hum. Servs.*, 993 F.2d 1525, 1528 (Fed. Cir. 1993).

Medical records and statements of a treating physician, however, do not *per se* bind the special master to adopt the conclusions of such an individual, even if they must be considered and carefully evaluated. Section 13(b)(1) (providing that "[a]ny such diagnosis, conclusion, judgment, test result, report, or summary shall not be binding on the special master or court"); *Snyder v. Sec'y of Health & Hum. Servs*., 88 Fed. Cl. 706, 746 n.67 (2009) ("there is nothing . . . that mandates that the testimony of a treating physician is sacrosanct—that it must be accepted in its entirety and cannot be rebutted"). As with expert testimony offered to establish a theory of causation, the opinions or diagnoses of treating physicians are only as trustworthy as the reasonableness of their suppositions or bases. The views of treating physicians should be weighed against other, contrary evidence also present in the record—including conflicting opinions among such individuals. *Hibbard v. Sec'y of Health & Hum. Servs.*, 100 Fed. Cl. 742, 749 (2011) (not arbitrary or capricious for special master to weigh competing treating physicians' conclusions against each other), *aff'd*, 698 F.3d 1355 (Fed. Cir. 2012); *Veryzer v. Sec'y of Dept. of Health & Hum. Servs.*, No. 06-522V, 2011 WL 1935813, at *17 (Fed. Cl. Spec. Mstr. Apr. 29, 2011), *mot. for review den'd*, 100 Fed. Cl. 344, 356 (2011), *aff'd without opinion*, 475 F. Appx. 765 (Fed. Cir. 2012).

The third *Althen* prong requires establishing a "proximate temporal relationship" between the vaccination and the injury alleged. *Althen*, 418 F.3d at 1281. That term has been equated to the phrase "medically-acceptable temporal relationship." *Id.* A petitioner must offer "preponderant proof that the onset of symptoms occurred within a timeframe which, given the medical understanding of the disorder's etiology, it is medically acceptable to infer causation." *de Bazan v. Sec'y of Health & Hum. Servs.*, 539 F.3d 1347, 1352 (Fed. Cir. 2008). The explanation for what is a medically acceptable timeframe must align with the theory of how the relevant vaccine can cause an injury (*Althen* prong one's requirement). *Id.* at 1352; *Shapiro v. Sec'y of Health & Hum. Servs.*, 101 Fed. Cl. 532, 542 (2011), *recons. den'd after remand*, 105 Fed. Cl. 353 (2012), *aff'd mem.*, 503 F. Appx. 952 (Fed. Cir. 2013); *Koehn v. Sec'y of Health & Hum. Servs.*, No. 11-355V, 2013 WL 3214877 (Fed. Cl. Spec. Mstr. May 30, 2013), *mot. for rev. den'd* (Fed. Cl. Dec. 3, 2013), *aff'd*, 773 F.3d 1239 (Fed. Cir. 2014).

B. *Legal Standards Governing Factual Determinations*

The process for making determinations in Vaccine Program cases regarding factual issues begins with consideration of the medical records. Section 11(c)(2). The special master is required to consider "all [ ] relevant medical and scientific evidence contained in the record," including "any diagnosis, conclusion, medical judgment, or autopsy or coroner's report which is contained

in the record regarding the nature, causation, and aggravation of the petitioner's illness, disability, injury, condition, or death," as well as the "results of any diagnostic or evaluative test which are contained in the record and the summaries and conclusions." Section 13(b)(1)(A). The special master is then required to weigh the evidence presented, including contemporaneous medical records and testimony. *See Burns v. Sec'y of Health & Hum. Servs*., 3 F.3d 415, 417 (Fed. Cir. 1993) (determining that it is within the special master's discretion to determine whether to afford greater weight to contemporaneous medical records than to other evidence, such as oral testimony surrounding the events in question that was given at a later date, provided that such determination is evidenced by a rational determination).

As noted by the Federal Circuit, "[m]edical records, in general, warrant consideration as trustworthy evidence." *Cucuras*, 993 F.2d at 1528; *Doe/70 v. Sec'y of Health & Hum. Servs.*, 95 Fed. Cl. 598, 608 (2010) ("[g]iven the inconsistencies between petitioner's testimony and his contemporaneous medical records, the special master's decision to rely on petitioner's medical records was rational and consistent with applicable law"), *aff'd*, *Rickett v. Sec'y of Health & Hum. Servs*., 468 F. App'x 952 (Fed. Cir. 2011) (non-precedential opinion). A series of linked propositions explains why such records deserve some weight: (i) sick people visit medical professionals; (ii) sick people attempt to honestly report their health problems to those professionals; and (iii) medical professionals record what they are told or observe when examining their patients in as accurate a manner as possible, so that they are aware of enough relevant facts to make appropriate treatment decisions. *Sanchez v. Sec'y of Health & Hum. Servs.*, No. 11–685V, 2013 WL 1880825, at *2 (Fed. Cl. Spec. Mstr. Apr. 10, 2013); *Cucuras v. Sec'y of Health & Hum. Servs.*, 26 Cl. Ct. 537, 543 (1992), *aff'd*, 993 F.2d at 1525 (Fed. Cir. 1993) ("[i]t strains reason to conclude that petitioners would fail to accurately report the onset of their daughter's symptoms").

Accordingly, if the medical records are clear, consistent, and complete, then they should be afforded substantial weight. *Lowrie v. Sec'y of Health & Hum. Servs*., No. 03–1585V, 2005 WL 6117475, at *20 (Fed. Cl. Spec. Mstr. Dec. 12, 2005). Indeed, contemporaneous medical records are often found to be deserving of greater evidentiary weight than oral testimony—especially where such testimony conflicts with the record evidence. *Cucuras*, 993 F.2d at 1528; *see also Murphy v. Sec'y of Health & Hum. Servs*., 23 Cl. Ct. 726, 733 (1991), *aff'd per curiam*, 968 F.2d 1226 (Fed. Cir. 1992), *cert. den'd*, *Murphy v. Sullivan*, 506 U.S. 974 (1992) (citing *United States v. United States Gypsum Co.*, 333 U.S. 364, 396 (1947) ("[i]t has generally been held that oral testimony which is in conflict with contemporaneous documents is entitled to little evidentiary weight.")).

However, the Federal Circuit has also noted that there is no formal "presumption" that records are accurate or superior on their face to other forms of evidence. *Kirby v. Sec'y of Health & Hum. Servs.*, 997 F.3d 1378, 1383 (Fed. Cir. 2021). There are certainly situations in which compelling oral or written testimony (provided in the form of an affidavit or declaration) may be more persuasive than written records, such as where records are deemed to be incomplete or inaccurate. *Campbell v. Sec'y of Health & Hum. Servs*., 69 Fed. Cl. 775, 779 (2006) ("like any

norm based upon common sense and experience, this rule should not be treated as an absolute and must yield where the factual predicates for its application are weak or lacking"); *Lowrie*, 2005 WL 6117475, at *19 ("[w]ritten records which are, themselves, inconsistent, should be accorded less deference than those which are internally consistent") (quoting *Murphy*, 23 Cl. Ct. at 733)). Ultimately, a determination regarding a witness's credibility is needed when determining the weight that such testimony should be afforded. *Andreu*, 569 F.3d at 1379; *Bradley v. Sec'y of Health & Hum. Servs*., 991 F.2d 1570, 1575 (Fed. Cir. 1993).

When witness testimony is offered to overcome the presumption of accuracy afforded to contemporaneous medical records, such testimony must be "consistent, clear, cogent, and compelling." *Sanchez*, 2013 WL 1880825, at *3 (citing *Blutstein v. Sec'y of Health & Hum. Servs*., No. 90–2808V, 1998 WL 408611, at *5 (Fed. Cl. Spec. Mstr. June 30, 1998)). In determining the accuracy and completeness of medical records, the Court of Federal Claims has listed four possible explanations for inconsistencies between contemporaneously created medical records and later testimony: (1) a person's failure to recount to the medical professional everything that happened during the relevant time period; (2) the medical professional's failure to document everything reported to her or him; (3) a person's faulty recollection of the events when presenting testimony; or (4) a person's purposeful recounting of symptoms that did not exist. *La Londe v. Sec'y of Health & Hum. Servs*., 110 Fed. Cl. 184, 203–04 (2013), *aff'd*, 746 F.3d 1334 (Fed. Cir. 2014). In making a determination regarding whether to afford greater weight to contemporaneous medical records or other evidence, such as testimony at hearing, there must be evidence that this decision was the result of a rational determination. *Burns*, 3 F.3d at 417.

C. *Analysis of Expert Testimony*

Establishing a sound and reliable medical theory often requires a petitioner to present expert testimony in support of his claim. *Lampe v. Sec'y of Health & Hum. Servs*., 219 F.3d 1357, 1361 (Fed. Cir. 2000). Vaccine Program expert testimony is usually evaluated according to the factors for analyzing scientific reliability set forth in *Daubert v. Merrell Dow Pharm., Inc.*, 509 U.S. 579, 594–96 (1993). *See Cedillo v. Sec'y of Health & Hum. Servs.*, 617 F.3d 1328, 1339 (Fed. Cir. 2010) (citing *Terran v. Sec'y of Health & Hum. Servs.*, 195 F.3d 1302, 1316 (Fed. Cir. 1999). Under *Daubert*, the factors for analyzing the reliability of testimony are:

> (1) whether a theory or technique can be (and has been) tested; (2) whether the theory or technique has been subjected to peer review and publication; (3) whether there is a known or potential rate of error and whether there are standards for controlling the error; and (4) whether the theory or technique enjoys general acceptance within a relevant scientific community.

Terran, 195 F.3d at 1316 n.2 (citing *Daubert*, 509 U.S. at 592–95).

In the Vaccine Program the *Daubert* factors play a slightly different role than they do when applied in other federal judicial settings, like the district courts. Typically, *Daubert* factors are employed by judges (in the performance of their evidentiary gatekeeper roles) to exclude evidence that is unreliable or could confuse a jury. By contrast, in Vaccine Program cases these factors are used in the *weighing* of the reliability of scientific evidence proffered. *Davis v. Sec'y of Health & Hum. Servs*., 94 Fed. Cl. 53, 66–67 (2010) ("uniquely in this Circuit, the *Daubert* factors have been employed also as an acceptable evidentiary-gauging tool with respect to persuasiveness of expert testimony already admitted"). The flexible use of the *Daubert* factors to evaluate the persuasiveness and reliability of expert testimony has routinely been upheld. *See, e.g.*, *Snyder*, 88 Fed. Cl. at 742–45. In this matter (as in numerous other Vaccine Program cases), *Daubert* has not been employed at the threshold, to determine what evidence should be admitted, but instead to determine whether expert testimony offered is reliable and/or persuasive.

Respondent frequently offers one or more experts in order to rebut a petitioner's case. Where both sides offer expert testimony, a special master's decision may be "based on the credibility of the experts and the relative persuasiveness of their competing theories." *Broekelschen v. Sec'y of Health & Hum. Servs*., 618 F.3d 1339, 1347 (Fed. Cir. 2010) (citing *Lampe*, 219 F.3d at 1362). However, nothing requires the acceptance of an expert's conclusion "connected to existing data only by the *ipse dixit* of the expert," especially if "there is simply too great an analytical gap between the data and the opinion proffered." *Snyder*, 88 Fed. Cl. at 743 (quoting *Gen. Elec. Co. v. Joiner*, 522 U.S. 146 (1997)); *see also Isaac v. Sec'y of Health & Hum. Servs*., No. 08–601V, 2012 WL 3609993, at *17 (Fed. Cl. Spec. Mstr. July 30, 2012), *mot. for review den'd*, 108 Fed. Cl. 743 (2013), *aff'd*, 540 F. App'x. 999 (Fed. Cir. 2013) (citing *Cedillo*, 617 F.3d at 1339). Weighing the relative persuasiveness of competing expert testimony, based on a particular expert's credibility, is part of the overall reliability analysis to which special masters must subject expert testimony in Vaccine Program cases. *Moberly*, 592 F.3d at 1325–26 ("[a]ssessments as to the reliability of expert testimony often turn on credibility determinations"); *see also Porter v. Sec'y of Health & Hum. Servs*., 663 F.3d 1242, 1250 (Fed. Cir. 2011) ("this court has unambiguously explained that special masters are expected to consider the credibility of expert witnesses in evaluating petitions for compensation under the Vaccine Act").

D. *Consideration of Medical Literature*

Both parties filed numerous items of medical and scientific literature in this case, but not all such items factor into the outcome of this decision. While I have reviewed all the medical literature submitted in this case, I discuss only those articles that are most relevant to my determination and/or are central to Petitioner's case—just as I have not exhaustively discussed every individual medical record filed. *Moriarty v. Sec'y of Health & Hum. Servs.*, No. 2015–5072, 2016 WL 1358616, at *5 (Fed. Cir. Apr. 6, 2016) ("[w]e generally presume that a special master considered the relevant record evidence even though he does not explicitly reference such evidence in his decision") (citation omitted); *see also Paterek v. Sec'y of Health & Hum. Servs.,* 527 F.

App'x 875, 884 (Fed. Cir. 2013) ("[f]inding certain information not relevant does not lead to—and likely undermines—the conclusion that it was not considered").

E. *Standards for Ruling on the Record*

I am resolving Petitioner's claim on the filed record. The Vaccine Act and Rules not only contemplate but encourage special masters to decide petitions on the papers where (in the exercise of their discretion) they conclude that doing so will properly and fairly resolve the case. Section 12(d)(2)(D); Vaccine Rule 8(d). The decision to rule on the record in lieu of hearing has been affirmed on appeal. *Kreizenbeck v. Sec'y of Health & Hum. Servs*., 945 F.3d 1362, 1366 (Fed. Cir. 2020); *see also Hooker v. Sec'y of Health & Hum. Servs*., No. 02-472V, 2016 WL 3456435, at *21 n.19 (Fed. Cl. Spec. Mstr. May 19, 2016) (citing numerous cases where special masters decided case on the papers in lieu of hearing and that decision was upheld). I am simply not required to hold a hearing in every matter, no matter the preferences of the parties. *Hovey v. Sec'y of Health & Hum. Servs*., 38 Fed. Cl. 397, 402–03 (1997) (determining that special master acted within his discretion in denying evidentiary hearing); *Burns*, 3 F.3d at 417; *Murphy v. Sec'y of Health & Hum. Servs.*, No. 90-882V, 1991 WL 71500, at *2 (Fed. Cl. Spec. Mstr. Apr. 19, 1991).

ANALYSIS

I. Petitioner Has Met His Prima Facie Burden of Proof for a Table Flu Vaccine-GBS Claim

The parties agree that Petitioner has alleged a Table claim in this case. Mot. at 11; Opp. at 7. GBS is listed as a Table injury for the flu vaccine, and thus a claimant seeking to meet its requirements must show (a) receipt of a covered form of the flu vaccine, (b) that the claimant did in fact experience GBS as defined in the Table's "qualifications and aids to interpretation," and (c) that onset (whether or not GBS could then be diagnosed, or was) occurred between three and 42 days after vaccination. 42 C.F.R. § 100.3.

All of these elements exist and/or have been satisfied in this case. There is no dispute that Petitioner received a covered version of the flu vaccine, and his GBS diagnosis is not contested. In addition, his onset occurred no sooner than October 10, 2020, since he reported weakness and tingling in his hands and feet having begun five days prior to his October 15th initial treater visit. Ex. 2 at 43. An onset occurring within five days of his October 5, 2020 vaccination falls within the timeframe set for a successful Table flu-GBS claim. Thus, the record preponderantly supports the *prima facie* elements of Petitioner's Table claim—meaning that Respondent can only prevail if he carries his shifted burden to prove a "factor unrelated." Section 13(a)(1)(B).

II. Respondent Has Not Established That Petitioner's CMV Infection Was the Likely Sole Substantial Factor in Causing Petitioner's GBS

A. *Legal Standards for "Factor Unrelated" Showing*

The nature of Respondent's "factor unrelated" burden in this case warrants explanation. While the evidentiary standard of preponderance applicable under *Althen* also applies in cases involving the shifted "factor unrelated" burden Respondent bears, governing authority has characterized this burden to be "higher"—or, more accurately, including an additional obligation not placed on claimants.

When a petitioner successfully establishes a *prima facie* case of causation, "the burden then shifts to the government to prove alternative causation by a preponderance of the evidence." *Cedillo,* 617 F.3d at 1338. The Vaccine Act defines "factors unrelated to the administration of the vaccine" to be matters "documented by the petitioner's evidence or other material in the record," such as "infection, toxins, trauma (including birth trauma and related anoxia), or metabolic disturbances which have no known relation to the vaccine involved, but which in the particular case are shown to have been the agent or agents *principally responsible for causing* the petitioner's illness, disability, injury, condition, or death." Section 13(a)(2)(B) (emphasis added).

As noted by the Court of Federal Claims in *Stone v. Sec'y of Health & Hum. Servs.*, 95 Fed. Cl. 233, 237 (2010),

> the standard for proving a "factor unrelated" is higher than the petitioner's burden of proving a prima facie case. Although a petitioner is required to show that the vaccine was a "substantial factor" in causing his or her injury, 'the petitioner need not show that the vaccine was the sole or predominant cause of her injury.' (*de Bazan,* 539 F.3d at 1351). The respondent's burden, by contrast, is to 'identify[ ] a particular [unrelated] factor (or factors) and present [ ] sufficient evidence to establish that it was the *sole substantial factor* in bringing about the injury.' *Id.* at 1354 (emphasis added). In order to prevail, therefore, the respondent must 'exclude[ ] the vaccine as a substantial factor.'

Id. Stone went on to observe that "[t]he difference between "substantial factor" and "sole substantial factor" is a meaningful one," noting that compensation could still be awarded even in cases where a factor unrelated had been shown to be substantial—but not "solely" substantial. *Stone,* 95 F.3d at 237 n.5 (citations omitted). This, in effect, describes what is sometimes referred to as a "*Shyface* analysis"—where two factors, including vaccination, are deemed potentially causal, but one cannot be found to predominate over the other. *Shyface v. Sec'y of Health & Hum. Servs.*, 165 F.3d 1344, 1352-53 (Fed. Cir. 1999).

In applying this standard, then, to the question of whether a factor unrelated explains an injury, special masters must conclude that the factor unrelated was the "sole substantial factor"—

although in so doing, the evidence deemed sufficient to reach that conclusion must only be *preponderant*, allowing the determination that it is "more likely than not" the factor unrelated was the sole substantial factor. That, in turn, leaves room for doubt (just as there is doubt in *any* preponderant determination that barely crosses the preponderant line). Thus, a finding that the vaccine has been excluded must only cross the "more likely than not" line, even if Respondent bears this obligation when petitioners never do. In practice, special masters have found the factor unrelated burden met by Respondent based on the same mix of evidence and weighing of items of literature versus expert testimony that they encounter when considering Petitioner's obligations.[15]

Importantly for present purposes, the strength of a claimant's prong one showing does not per se make it *less likely* that the proposed factor unrelated was not the sole substantial factor (although the evidence offered pro and con must still be weighed, and therefore evidence relevant to a claimant's initial showing will in many cases get additional airing while performing the burden-shifted analysis). And I am aware of no case law suggesting that once a claimant offers preponderant evidence sufficient to meet the Table elements for a claim, that showing somehow disadvantages Respondent in attempting to prove factor unrelated. The concession of causation implicit in a Table claim does not mean the relevant vaccine can be assumed to have played *some* role in causation, regardless of the role the factor unrelated is demonstrated to have played.

B. *Nature of CMV Infection*

CMV is a herpes virus with an estimated seroprevalence of 45%-100% in the general, worldwide population. R. Lachmann et al., *Cytomegalovirus (CMV) Seroprevalence in the Adult Population of Germany*, 13 PLOS One 1,1 (2018), filed as Ex. 51 (ECF No. 49-14) ("Lachmann"). Primary CMV infection usually occurs during childhood and early adolescence, after which the virus establishes a latent phase mainly within leukocytes. Al-Omari at 1. Transmission of the virus can occur through contact with CMV-infected body fluids during primary infection or reactivation. Lachmann at 1. Although CMV infections are typically asymptomatic in immunocompetent hosts, they can cause life-threatening complications in immune-compromised individuals. *Id.*

C. *Respondent Has Not Carried his Shifted Burden*

The parties' experts agree that Petitioner tested positive for CMV while being treated for GBS (even if they disagree as to the precise severity of the infection). They further concur that GBS frequently is attributable to an antecedent infection. And it is also likely the case – as preponderantly established by Respondent – that a broad array of infections are *more likely*

[15] To give one example, after remand of the *Stone* case (where Respondent maintained that a child's Dravet syndrome was solely due to a genetic mutation rather than vaccination), the special master was readily able to conclude under the proper standard that Respondent had met his burden, relying on a showing that included (a) the determination to give more weight to Respondent's expert testimony than Petitioner's, (b) the highly persuasive evidence of the alternative cause, and (c) an absence of record evidence that the vaccine *itself* had caused any harm to the child's brain (as would needed to have been shown to conclude the vaccine caused injury in accordance with the theory alleged). *Stone v. Sec'y of Health & Hum. Servs.*, No. 04-1041V, 2011 WL 836992. at *3 (Fed. Cl. Spec. Mstr. Jan. 20, 2011), *mot. for review den'd*, 99 Fed. Cl. 187 (2011), *aff'd,* 676 F.3d 1373 (Fed. Cir. 2012).

explanatory of GBS than *any* vaccine. First Collins Rep. at 7. But Respondent must still demonstrate that in this case, the established CMV infection was *itself* the sole causal factor of Petitioner's GBS.

Respondent has unquestionably succeeded in establishing several disputed issues in his favor. It has been preponderantly established, for example, that a CMV infection "can cause" GBS. *See e.g.*, Yuki at 1. I also find persuasive Dr. Collins's contention that medical science does not require a CMV infection to be clinically obvious, and/or especially virulent, with unmistakable or severe symptoms for it to cause GBS. Special masters routinely find a vaccine caused a subsequent injury based on records containing little to no evidence of *any* post-vaccination reaction – and it is therefore just as possible that a latent infection with an almost subclinical course could equally result in disease later. And correspondingly, the fact that a CMV infection reflects a reactivation, rather than an acute/initial infectious process, does not undermine the capacity of the infection to cause GBS.

The parties also disagree as to the relevance of Petitioner's own infectious severity. And on this point, the evidence is in equipoise. On the one hand, it cannot be disputed that Petitioner had no CMV infection symptoms at all before either onset of his GBS or discovery of the infection through testing. Certainly, it was not a severe infection with obvious manifestations. But the significance of this fact is unclear. Dr. Collins did persuasively establish that severity of infection is not a prerequisite to GBS's development – and she also successfully demonstrated that the risk from a merely reactivated infection was not necessarily less than that from a primary infection. At the same time, Petitioner's infectious work-up did not deem the infection particularly significant – although that work-up was clearly cursory and did not at all delve into the possible relationship between the infection and Petitioner's GBS.

Ultimately, I do not accept Petitioner's effort to give great weight to the severity of the infection in resolving whether the infection could have caused his GBS. But I nevertheless still do not find Respondent has carried his "factor unrelated" burden – despite his other successes. For on this record, I cannot conclude that preponderant evidence establishes that Petitioner's CMV infection likely "did cause" his GBS – the second *Althen* prong (which applies when evaluating Respondent's factor unrelated showing). *See Knudsen*, 35 F.3d at 549.

The record simply does not reveal enough about Petitioner's personal health circumstances to conclude that the discovered CMV infection likely *was* the reason he developed GBS – even if in *most cases* such an infection would have more explanatory value than receipt of the flu vaccine. Even if I ignore the question of infectious severity, there is nothing from the medical record that suggests *in this context* that the infection was causal. Rather, the record presents evidence that an infection (which may or may not have existed at the time of vaccination, or began after, or reflected an unexplained reactivation) *merely existed*. I fully accept Respondent's contention that a CMV infection could cause GBS generally, and even that it is more likely to result in GBS than a flu

vaccine. But I would require additional corroborative proof to find that *in this case*, it did so – and that evidence is lacking.

Dr. Collins's careful reading of the testing results does not change my analysis. All she has done is show results – the liver functioning or lymphocyte – *corroborating* the CMV infection. This does not make it more likely the infection *did cause* GBS (even if some items of literature show instances in which it was determined that a CMV infection likely caused GBS because of the results of those tests). Simply because the infection was *present* around the time of Petitioner's onset does not support the conclusion that it also likely caused those initial GBS-oriented symptoms as well. And her arguments about the significance of certain results, or Petitioner's clinical manifestations and what they say about the best explanation for Petitioner's GBS, were ultimately unavailing.

In finding as I have, I stress the fact that the medical record does not allow me to conclude it "more likely than not" that the CMV infection explains Petitioner's GBS. In effect, Respondent is arguing that the established fact of the infection, and its high association with GBS when compared to the flu vaccine, makes it the "only" possible cause. But I routinely reject that kind of logic when offered by petitioners in causation-in-fact cases, where the burden has not shifted.

Thus, a silent or incomplete record that shows no evidence of a vaccine reaction, or other testing results consistent with some vaccine-associated process, can result in denial of entitlement on the second *Althen* prong. *See, e.g., Bender v. Sec'y of Health & Hum. Servs.*, No. 11-693V, 2018 WL 3679637, at *34-35 (Fed. Cl. Spec. Mstr. July 2, 2018) (record did not support finding that a Hepatitis A vaccine did cause a claimant's TM, given absence of proof of record corroboration of vaccine-instigated process), *mot. for review den'd,* 141 Fed. Cl. 262 (2019). Indeed, I frequently call petitioners and their experts to task for assuming that the required "logical sequence of cause and effect" is demonstrated merely by the *temporal* association between a vaccine's administration and subsequent illness or other injury. *See e.g., M.R. v. Sec'y of Health & Hum. Servs.*, No. 16-1024V, 2023 WL 4936727, at *34 (Fed. Cl. Spec. Mstr. June 30, 2023) ("At bottom, the 'best' evidence of a vaccine-injury association that can be mustered is the fact that [the injury] occurred within a week of vaccination. But without other proof, that kind of temporal showing is not and has never been enough of a basis for a favorable entitlement decision" (citing *Grant*, 956 F.2d at 1148)). Claimants do not get to breeze past *Althen* prong two once they show a vaccine could be causal of a specific injury.

I have also found for Respondent in cases involving the factor unrelated analysis – but those results underscore what is absent in this case. *See, e.g., White v. Sec'y of Health & Hum. Servs.*, No. 20-1319V, 2023 WL 4204568 (Fed. Cl. Spec. Mstr. June 2, 2023), *mot. for review den'd*, 168 Fed. Cl. 660 (2023), *appeal docketed*, No. 24-1372 (Fed Cir. Jan. 23, 2024). In *White*, as here, I determined that the Table elements of a claim for GBS after receipt of the flu vaccine had been met – leading Respondent to attempt to carry his shifted, "factor unrelated" burden. *White*, 2023 WL 4204568, at *15. The record in *White* also revealed that the claimant had an

obvious *H. influenzae* infection[16] that could have been causal. *Id.* at *17. But that infection not only required treatment due to its virulence, but could be shown as having begun *after* vaccination while also *closer in time* to Petitioner's GBS onset. *Id.* at *18. Here, by contrast, I cannot ascertain when Petitioner's CMV infection began – and even if a reactivated infection can still cause GBS (something I find Respondent did establish), I am unable to find the infection was primary or reactivated. In addition, treaters in *White* agreed the infection was a good etiologic explanation for his injury. *Id.* at *17. Such facts relevant to the prong two analysis are absent in this case.

The foregoing is not impacted by Respondent's strong success in associating the CMV infection with GBS. This is because a claimant's obligation to submit sufficient preponderant evidence on the "did cause" *Althen* prong cannot be ignored or deemed subsumed within a claimant's success on the first prong. The same is true for the parties' debate over the relative risks of vaccination versus a CMV infection. Respondent has shown not only that infections in most cases are better associated with GBS than vaccination, but also that a CMV infection is *itself* a risk factor for GBS. *See e.g.*, Yuki at 1 ("Cytomegalovirus is the most common viral antecedent infection [to GBS]"). But this does not mean that when both factors are present, that the better-associated cause *was* the likely, or sole, cause. *Shyface* itself rejects that reasoning, allowing for the possibility that a vaccine could always be an important causal factor *even if* another causal factor exists, and/or is more likely causal in most circumstances. *Shyface*, 165 F.3d at 1353. That determination does not depend on absolute causal risks, outside the context of the facts of a specific case.[17] Rather, the medical record relevant to a claimant's unique circumstances bears heavily on resolution of the question – and here, that record is too silent on the actual likely cause of Petitioner's GBS to conclude it could *only* have been the infection.

This case also does not turn on which expert was more persuasive overall, or whose marshalling of the evidence was the most successful. Dr. Collins made many effective and persuasive contentions about the CMV infection and its general association with GBS. Dr. Kelesidis, by contrast, strained to demonstrate the comparative risk of the flu vaccine as equivalent, and did not otherwise undermine Respondent's contentions that a CMV infection could cause GBS even if the infection was only a reactivation. But I simply cannot discern on this record that the CMV infection "more likely than not" caused Petitioner's GBS. The propensity of the flu vaccine to cause GBS really does not weigh one way or another on this determination.

[16] In *White*, the severity of the infection, and obvious evidence of its clinical presence, proved particularly relevant – and this, coupled with the fact that the infection was (all things being equal) more likely to cause GBS, resulted in a factor unrelated finding for Respondent. Here, as noted above, I do not give great weight to the absence of clinical evidence of a CMV infection (and it certainly does not suggest a CMV infection is *less likely* to cause GBS if the infection is not active or virulent). But it remains the case that the lack of objective evidence of the infection's impact on Petitioner bears somewhat on analysis for the second *Althen* prong.

[17] Indeed, in almost any vaccine injury case, it is far more likely other factors can cause an injury in question than the vaccination the claimant received. But just as the general safety of vaccines for most of the population is not relevant to whether a specific claimant was harmed, the low likelihood of a vaccine causing the injury in absolute terms – and when compared to other known causes – does not mean it could not have caused the injury *to the specific claimant*.

In the end, Respondent did not fail in proving factor unrelated because he could not exclude the vaccine as causal. I do not even reach that question herein – and the existence of the Table claim of GBS after receipt of the flu vaccine does not mean that *in every case* where such a vaccine has been administered, the flu vaccine should be *assumed* to have played some role in causing GBS subsequent to it. Rather, Respondent's inability to prove that CMV in this case "did cause" Petitioner's GBS was dispositive.[18]

CONCLUSION

Petitioner has prevailed in his Table claim. A damages order will follow this Decision.

IT IS SO ORDERED.

/s/ Brian H. Corcoran
Brian H. Corcoran
Chief Special Master

[18] Admittedly, this matter presents a very close evidentiary determination, with evidence pro and con on the disputed "did cause" element. In such circumstances, Program case law counsels deciding the issue in a petitioner's favor. *Althen*, 418 F.3d at 1280. Even though a CMV infection *clearly can cause GBS* – and likely does so far more often than the flu vaccine – I cannot find sufficient evidence that it likely did so *here*, given the ambiguous nature of the demonstrated infection and its impact on Petitioner's health.